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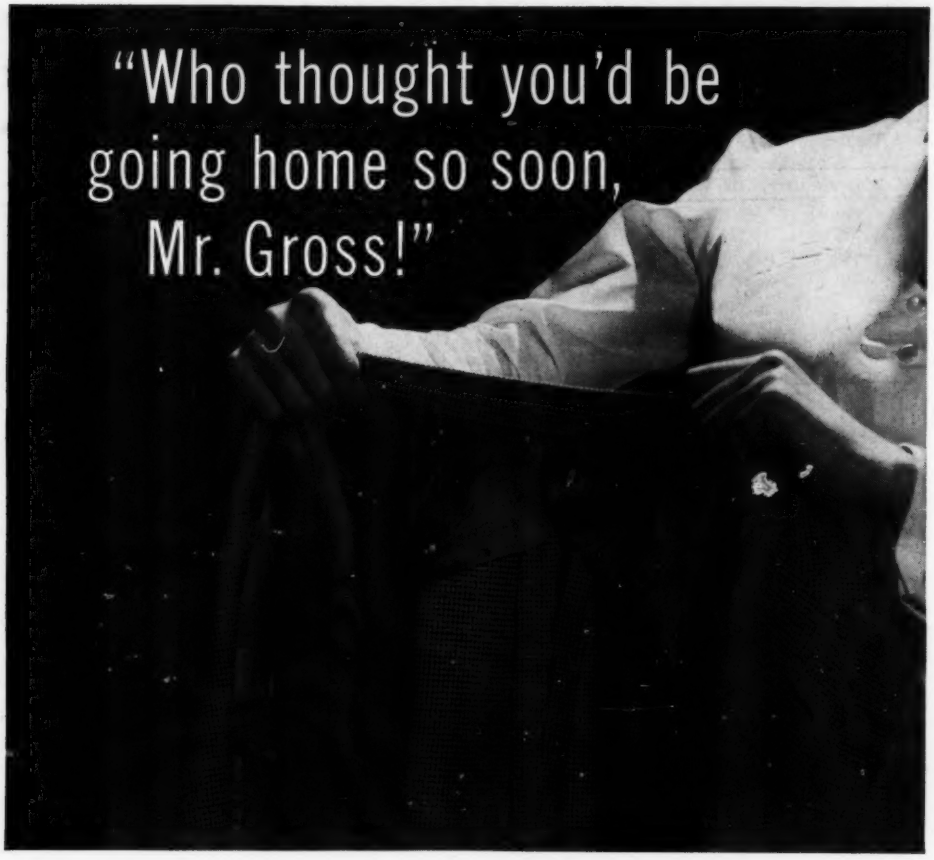

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1. Fox, C. L. Jr., et al.: An Electrolyte Solution Approximating Plasma Concentrations with Increased Potassium for Routine Fluid and Electrolyte Replacement, J. A. M. A., March 8, 1952.

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De Rebus Medicis Et Politicis

BY ROBERT B. HOMAN, JR., M. D., EL PASO, TEXAS
MEMBER OF THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

A RESOLUTION FROM THE AMERICAN LEGION

At various intervals this column has called your attention to the problem of medical care of the veterans of our wars, to the program of the Veterans Administration under laws enacted by Congress, and to the consideration of and action by the House of Delegates of the A. M. A. in respect to this problem. It is logical, therefore, for you to have before you a resolution recently passed by the American Legion in convention. This resolution was presented to said convention by the Legion's Rehabilitation Committee and was passed by the convention without debate.

"WHEREAS, the hierarchy of certain uninformed and ill advised medical organizations of the United States recently offered testimony before the Subcommittee on Hospitals of the Committee on Veterans Affairs of the House of Representatives, 83rd Congress 1st Session as follows: 'We do not feel that a Veteran, per se, who has suffered no disability by reason of his service in the Armed Forces, who has come out well and whole, has any more demand on the Federal Government than any other citizen. He has discharged the normal obligation of a man to bear arms in the defense of his country. He should do that as a part of his citizenship, and by virtue of it, unless his service has resulted in some disability, we do not feel that he has any call on the Federal Government. We think that if he is indigent, he is a community responsibility.' And

"WHEREAS, The same medical organizations have gone on record at their recent National Conventions that it is their belief that these organizations must concern themselves not with the efficacy of the Veterans Administration in the administration of enabling legislation but rather with the broad question of whether such legislation is sound,

"NOW, THEREFORE BE IT RESOLVED, By the American Legion in National Convention assembled in St. Louis, Missouri, this 2nd day of September, 1953, that we here-by reiterate our position of many years that the man or woman who was called into the Armed Forces of this Country by our Government, told where and when to go, what to wear and how, what to eat, when to go to bed and when to arise, what shots to take, and who, for risking his or her life, has been placed in a very special class by that Government, and

"BE IT FURTHER RESOLVED, That we know that historically, the citizens of the United States, through our Congress, have always considered all Veterans a special class of citizens and have always shown the respect and affection for these Veterans by certain benefits and just compensations, and

"BE IT FURTHER RESOLVED, That we reiterate and again support and endorse the basic principle of our Country that Veterans who have fought for and defended all of our citizens are indeed a distinct class and the direct and specific responsibility of our Federal Government when in need, or are sick, broke and have no place to go."

If the majority of Veterans of our past wars support such a resolution in their personal thinking, we are going to need many more beds in Veterans facilities, many more nurses and technicians than we now have. The voluntary hospital system cannot compete against the government for trained personnel. This should be a matter of concern to all Americans, veterans and non-veterans alike.

AIR POLLUTION

Air Pollution: A National Problem
Marin, R. B., J. Med. Soc. New Jersey 11:464, 1952

Smoke control in urban areas is only part of the problem. Pollution by fly ash, soot, nitrogenous substances, sulfur oxide, hydrocarbons, aldehydes, acids and radio-active contaminants must be considered. Over 50,000 tons of various substances are discharged daily into the air over Los Angeles County. Every month 79 tons of grit and grime are deposited per square mile in the New York-New Jersey metropolitan area. "How such huge daily atmospheric chemical reaction affects humans is a matter of serious conjecture."

Clinical Clippings, January, 1953.

APHORISMS AND MEMORABILIA TRUTHS AND CONCEPTS CONCERNING THE CARDIO-VASCULAR SYSTEM

By Andrew M. Babey, M. D., Las Cruces, N. M.

1. "After the calf, the intrinsic muscles of the foot are those most often affected. The pain is brought on by walking, but it is less cramp-like than calf pain and it is often described as the sensation felt when "crossing pebbles barefoot"; this is because the ischaemia affects not one large muscle, but a number of small muscles which suffer short periods of claudication in turn, and so give rise to this "knobbly" pain. It is quickly relieved by rest. In advanced disease any muscle may claudicate, and pain on exercise has been described in the thigh, gluteal and lumbar muscles. Except in advanced thromboangiitis, it is rare in the upper extremity." —Sir James Learmonth, K. C. V. O., and A. J. Slessor, M. V. O.; Pain and Its Problems, —Blackeston and Co. 1950 *p* 50.

2. "In most cases, the site of claudication indicates the approximate level of the arterial obstruction; if the foot alone claudicates, the obstruction is in the lower leg, whereas if the calf is affected, a popliteal or femoral occlusion will be suspected. In most cases, palpation of the pulses will confirm these suspicions, but occasionally claudication occurs in the calf when distal pulses are present. In this event, the arterial occlusions lie in the sural arteries which supply the two heads of the gastronemius. These are end-arteries, and collateral circulation is not available to meet the needs of exercise." —Learmonth and Slessor, *loc. cit. pp.* 50-51.

3. "Rest pain is constant, and is not relieved by recumbency. Indeed it is most severe at night, keeping the patient awake. It is usually situated in the toes and metatarsal region, but in severe cases may involve the foot and lower leg. In character, it is a gnawing, aching pain, to which hot, burning sensation may be added; it causes extreme distress, and even partial relief is hard to obtain, but not infrequently the patient finds that rest pain is more easily tolerated when the feet are dependent, and he may spend hours at night with the feet dangling over the bedside, or he may attempt to sleep in a chair." — Learmonth and Slessor, *loc. cit. pp.* 52.

4. "Rest pain may be an indication that gangrene is imminent; there are few patients who, having been forced to attempt sleep in a chair, have preserved their limbs for any length of time. However, not all limbs progress to gangrene, and with rest and care, particularly avoidance of even minimal injury, a number may be nursed back to some degree of usefulness, which will be directly proportional to the extent of collateral circulation which can be fostered and maintained." — Learmonth and Slessor *loc. cit. pp.* 52.

5. "Rest pain is the result of severe ischaemia of tissues and of sensory nerve endings. When the latter suffer from ischaemia, the more highly specialized are affected first, as can be demonstrated objectively by testing skin sensitivity. Light touch and light pinprick will not be appreciated in a zone of varying extent, but with proximal margin always circular; the anaesthetic area is of boot, shoe or slipper distribution, or it may be smaller, involving only the forefoot or toes. It will invariably be found that crude forms of sensation, such as deep pinprick or heavy pressure, are appreciated within a territory in which light touch and light pinprick are not; this conforms to the varying vulnerability of different types of sensory nerve endings. The character of rest pain can now be understood: The dull, hot pain is that initiated by the most crude types of end-organs, the only ones to survive the ischaemia. That tinglings are felt in the prodromal phase is due to the milder degree of the ischaemia, which has not yet inactivated the more sensitive end-organs." — Learmonth and Slessor, *loc. cit. pp.* 53.

6. "While the extent of the pericardial disease was reflected in the number of leads affected, the degree of T wave inversion told of the penetration of the pericardial infection into the myocardium. This was tested as a means of predicting the difficulty met with in stripping the thickened pericardium from the heart; thus, in 10 out of 11 cases in which the changes in the T wave were of a light character the pericardium peeled easily from the heart at operation, compared with the more difficult task in patients who showed depe T wave inversion." — William Evans and Frederic Jackson, *British Heart Journal, Jan.* 1952 *pp.* 58.

7. "The most characteristic distinguishing feature of the oesophagram of constrictive pericarditis was the presence of a relatively clear area between the oesophageal

stream and the denser part of the cardiac shadow, whereas in mitral stenosis these two structures usually lay contiguous one with the other. This difference is explained by the displaced undistended left auricle in constrictive pericarditis and by the distended left auricle in mitral stenosis." — Evans and Jackson, *loc. cit. pp.* 60.

8. "In patients where constrictive pericarditis has lasted a longer time a knowledge of the extent of the liver damage is very desirable for it may be so great as to predict failure of the procedure of cardiac decompression as a means of relieving the symptoms, and ascites may persist as the direct outcome of the hepatic fibrosis." — Evans and Jackson, *loc. cit. pp.* 64.

9. "In regard to the barbiturates I should like to point out that a recent study on the use of sodium amytal was notable in that decreased cardiac output resulted when the drug was used." — Dr. Henry Schroeder, *American Journal of Med. Jan.* 1952 *pp.* 103.

10. "Cyanosis, usually seen in the ears, nose, lips, and fingers, was present in half the cases, and was more conspicious in those patients in whom the condition was known to have produced symptoms for two or more years. It was absent in the early cases and this should never by itself oppose a diagnosis of constrictive pericarditis." — William Evans and Frederic Jackson, *British Heart Journal, Jan.* 1952 *pp.* 56.

11. "Distention of the neck veins is a constant physical sign in constrictive pericarditis and it was met with in each of our cases." — Evans and Jackson, *loc. cit.*

12. "Since dysponea at rest is uncommon in constrictive pericarditis, the presence of distended neck veins in a patient who is able to lie comfortably in the recumbent posture is a characteristic feature of the disease."— Evans and Jackson, *loc. cit.*

13. "Hepatic enlargement was present in each of the 30 patients, and this physical sign ranks with distended veins as a valuable aid to diagnosis; indeed, if these two signs are missing, the diagnosis of constrictive pericarditis should not be entertained."— Evans and Jackson, *loc. cit.*

14. "Pulmonary crepitations were absent on auscultation except in those where the lung showed fibrosis, probably tuberculosis in origin. Further, pulmonary congestion was not found on cardioscopy. Absence of such pulmonary signs in a patient thought to have heart failure should turn attention to the diagnosis of pericardial disease." — Evans and Jackson, *loc. cit. pp.* 57.

15. "Evidence of cardiac enlargement was never present on clinical examination and the cardiac impulse was usually quiet and often absent; the apex beat when it could be located was never displaced outwards." — Evans and Jackson, *loc. cit.*

16. "Unlike most instances of right heart failure constrictive pericarditis does not give rise to significant murmurs. Indeed, should a case exhibit an obvious murmur a diagnosis of the pericardial lesion is unlikely to be the true one." — Evans and Jackson, *loc. cit.*

17. "The Medical Research Council of England recommends that for the treatment of endocarditis, regardless of how sensitive the offending organism may be, the minimal daily dose of penicillin should be 2,000,000 units, and the period of treatment four to six weeks."—J. Geraci (Mayo Clinic) *Minn. Med. Sept.* 1952 *p.* 861.

18. There are cases that I have followed now for 25 or 30 years since their original attack of angina pectoris or following coronary occlusion with myocardial infarction. There are patients whom I am now following who are fairly well 25 or more years after a severe myocardial infarct or after angina pectoris which subsided. Hence, coronary heart disease sufficient to cause symptoms may be very long-standing. — Dr. Paul White, *Am. Practitioner. Sept.* 1952, *p.* 750.

19. "I believe that the most dangerous cases of coronary heart disease are not those heralded by myocardial infarction, but those who have angina pectoris decubitus with normal electrocardiograms and who are therefore considered safe. We see many patients with angina decubitus whose hazards have been underestimated." — Dr. Paul White, *loc. cit.*

20. "I am glad that Dr. Aub told the story of Sir James Mackenzie. He would have angina pectoris walking up to the golf links, but when he got there he was interested in the game, got his second wind and played golf very well. This is very commonly true especially among young people who have only a mild amount of coronary insufficiency. They may have an attack on the first hole, but the 17 remaining holes they can take care of all right. That, however is not good enough. They should not play golf at all until they can play the first hole without symptoms."—Dr. Paul White, *loc. cit. p.* 751.

(To Be Continued)

SOUTHWESTERN MEDICAL MEETING IN TUCSON, OCTOBER 29 THROUGH 31

An attractive scientific and social program has been arranged for the 35th annual meeting of the Southwestern Medical Association in Tucson, Arizona, October 29 through 31, and the Convention Committee, headed by Dr. Harold Kohl, is preparing for a large attendance of physicians who in past years have been rotating between the cities of Albuquerque, El Paso and Phoenix for the annual gathering.

The following list of speakers and subjects have been announced: —

1. EDMUND SPAETH, M. D.—Professor of Ophthalmology, University of Pennsylvania Graduate School of Medicine, Philadelphia.
   Subjects to be announced.
2. MERL J. CARSON, M. D.—Professor of Pediatrics, University of Southern California School of Medicine, Los Angeles, California.
   Subjects to be announced.
3. MARION B. SULZBERGER, M. D.—Professor of Dermatology and Syphilology, New York University Postgraduate Medical School, New York.
   Subjects to be announced.
4. WILLIAM PAUL THOMPSON, M. D.—Clinical Professor of Medicine, College of Medical Evangelists, Los Angeles.

   Subjects:

   1. Selection of Patients for Mitral Commissurotomy.
   2. Prevention of Coronary Thrombosis.
   3. Management of Congestive Heart Failure.
5. DONOVAN C. BROWNE, M. D.—Associate Professor of Medicine, Tulane University, New Orleans.

   Subjects:

   1. Controversial Problems in Management of Peptic Ulcer.
   2. Post Gastrectomy Dumping Syndrome.
   3. Management of Chronic Ulcerative Colitis.
6. HENRY K. RANSOM, M. D.—Professor of Surgery, University of Michigan Medical School.

   Subjects:

   1. Diverticulitis of the Colon.
   2. Surgical Treatment of Duodenal Ulcer.
   3. Diseases of the Breast.

Headquarters for the meeting will be the Pioneer Hotel. C. Edgar Goyette, manager of the Tucson Chamber of Commerce, is working with convention officials in making hotel reservations.

Other meetings to be held during the convention include the regional meeting of the American College of Physicians, the Southwestern Dermotology Society meeting, the regional meeting of the Southwestern Surgical Society and the Arizona Society of Internal Medicine.

Social events include the football game between the University of Arizona and West Texas State on October 31, a golf tournament on October 31 and a dinner dance on the evening of October 29, for which dress is optional.

Officers of the Southwestern Medical Association are Dr. Wesley O. Connor, Jr., of Albuquerque, president; Dr. Willard W. Schuessler of El Paso, president-elect; Dr. Harold Kohl, Tucson, first vice-president; Dr. John H. Dettweiler, Albuquerque, second vice-president; Dr. Leslie B. Smith, Phoenix, third vice-president; and Dr. Celso C. Stapp, El Paso, secretary-treasurer.

Officers of the Pima County Medical Society, which is host for the convention, are Dr. W. R. Manning, president, Dr. Donald F. Hill, vice-president, Dr. W. B. Steen, secretary-treasurer.

Convention committee members working under the direction of Dr. Kohl are Dr. N. K. Thomas, program chairman, Dr. H. D. Cogswell, chairman of the arrangements committee, Dr. H. J. Rowe, publicity chairman, Dr. D. L. Secrist, football game, Dr. D. J. Heim, chairman of the finance committee, Dr. E. R. Updegraff, golf, Mrs. Leo Kent, chairman of women's activities, and Mrs. Darwin W. Neubauer, chairman of the dinner dance committee.

COMMENTS ON THE TREATMENT OF REPTILE POISONING IN THE SOUTHWEST

By Frederick A. Shannon, M. D., Wickenburg, Arizona

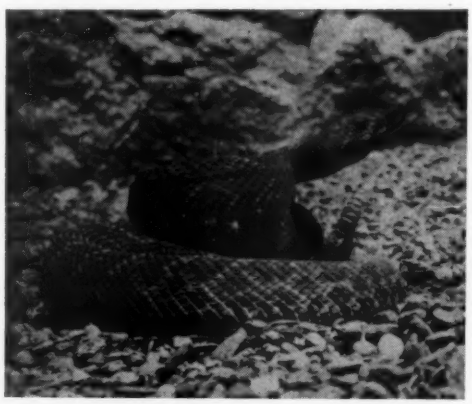
This article was undertaken with some hesitancy, as there are a number of good papers on the treatment of snakebite in the existing literature, such as those of Gloyd[1] and Stickel.[2] However, one of the most significant contributions, a paper by Clifford H. Pope entitled "The snake-bite problem," was published in a comparatively obscure journal,[3] and Pope's conclusions have only rarely reached medical journals. In addition, the author feels that a written defense of those doctors who choose to omit antivenin from their therapy is advisable for medicolegal reasons. Finally, the author has felt that a checkrein should be applied to certain dangerous and apparently unsubstantiated publicity given to cold treatment, or "cryotherapy," for snake-bite.

SNAKE-BITE PROBLEM IN THE SOUTHWEST

The problem of snake-bite assumes complex proportions in the Southwest. Distances are great between towns, and the patient is frequently far away from help when bitten. Thus considerable time may elapse before he can present himself for treatmnt.

General discussion of species: The bewildering variety in species of snakes raises the question of whether a bite is poisonous or non-poisonous. Of the fifty-odd species and sub-species of poisonous snakes in the United States, at least 40 occur in the Southwest. Of these, fortunately, 14 or more are rear-fanged snakes, and their bite may not break the skin, due to their small size. If they do succeed in breaking the skin, the venom is of low potency and not likely to be dangerous to the life of a human victim. Nevertheless, a bite from a rear-fanged snake may be misleading to the physician, as the patient may experience considerable temporary pain. Objective evaluation of the victim will reveal that there is no marked systemic reaction, although considerable emotional involvement may be present. Swelling in the involved extremity is also minimal. The two dozen or so dangerous subspecies and species are all pit vipers (Family Crotalidae), with the exception of two different species of coral snakes belonging to the Family Elapidae.

Of these, *Micrurus fulvius tenere* enters the area of discussion only in eastern Texas. Although not aggressive, the snake takes advantage of its liking for moist habitats by having an annoying habit of showing up on summer evenings in the damp grass of freshly watered lawns. One was taken under these conditions well within the city limits of San Antonio. The other coral snake species, *Micruroides euryxanthus*, is found in the deserts of southern Arizona, whence it extends north in the valleys of the low basin-and-range topography approximating the Colorado River, possibly as far as extreme southwestern Utah. This species, which is also nocturnal, likewise has an affinity for freshly watered lawns. In the absence of better criteria, the Arizona coral snake should be rated as less dangerous than the Texas species, as it reaches a maximum length of less than 20 inches, compared to a maximum of nearly twice as long for the Texas coral snake. Both species are slender, small-headed snakes, with banded red, yellow or cream, and black, and both may be easily differentiated from harmless species resembling them by the fact that coral snakes have the red color adjacent to the yellow, whereas the red is adjacent to the black on king snakes. "Red against yellow kill a fellow; red against black venom lack."



Western Diamond-Back Rattlesnake

Southwestern pit vipers include the closely related copperhead and water moccasin

as well as the rattlesnakes. The former two snakes are found only in eastern Oklahoma and Texas, but rattlesnakes are found throughout the Southwest, Arizona having no less than 16 species and subspecies. Of these, the Western diamond-back is probably the most dangerous because of its large size and ugly disposition. In eastern Texas and Oklahoma, physicians will not infrequently be called upon to treat bites inflicted by harmless water snakes superficially resembling the moccasin or copperhead. One would think that there would be no danger of confusion in the rest of the Southwest, because any snake, with the exception of the coral, is harmful if it bears a rattle, harmless if it doesn't. However, the author has received numerous bull snakes for his collection of specimens from persons confusing them with diamond-backs. There is no need to go into a taxonomic discussion of the different poisonous snake species, as there are a number of excellent source books on the subject. Dr. William Stickel, in a leaflet of the Fish and Wildlife Service,[3] has a short summary with range diagrams of the poisonous snakes of the United States, as well as an outline of treatment.

CLASSIFICATION AND PATHOLOGICAL ACTION OF VENOM TYPES

There are two basic venom types, that of the pit vipers which is composed largely of proteolytic, hemolytic, coagulant, and anticoagulant factors, and that of the coral snakes which is neurotoxic. This differentiation should not be drawn too finely, as there are components of both in all snake venoms, and relative concentrations vary from species to species. Venom composition is poorly understood due to the complexities of the protein molecular structures. Porges[4] notes that the phosphatidases play a prominent part in poisoning. Thus lecithinase acts upon lecithin causing hydrolysis and yielding, among other factors, oleic acid and lysolecithin. Lysolecithin, like lecithin, is actively hemolytic, and its effects on the heart are similar to those of venom. It acts upon isolated heart muscle and causes contractures, fibrillation, loss of potassium, and excitability. By attacking capillary endothelium it may cause hemorrhage of the lungs. Phosphatidase action in forming lysolecithin is accompanied by the liberation of histamine, which may result in an abrupt fall of systemic blood pressure. According to Stanley,[5] a pit viper-bite will produce a moderate leukocytosis of 10-12,000 with a normal differential. Erythrocyte destruction may be gross, with hemolytic anemia, a prolongation of bleeding and clotting time, increased capillary fragility, hemoglobinuria, and albuminuria. The latter is due in part to the histolytic effect of the venom on the capillaries and basement membrane of Bowman's capsule. Local and later often more generalized destruction of capillaries and protein loss intercellularly with resulting loss of vascular osmotic pressure are undoubtedly major causes of the intense swelling and edema resulting from a pit viper bite. Laryngeal and pulmonary edema may result from a serious bite, as may purpura so severe that mucus and mucocutaneous junctions may bleed extensively in addition to extravasatins of blood into subcutaneous and cutaneous tissues. Loose clumping of destroyed blood cells may result in embolic dissemination to any part of the body. Agglutinin and agglutinogen destruction may be so severe that blood typing is impossible. Profound shock may result from blood loss, from alteration of proteins, or occasionally, especially in cases of previous snake bite, from anaphylaxis. The patient may lose consciousness and become incontinent, neither criterion necessarily associated with fatality. Hyaluronidase activity is high in some venoms, causing an increase not only in their own invasive powers, but in those of any bacteria which may be introduced into the wound.

The neurotoxic venom of the coral snakes does not have the sledge hammer effects of the hemotoxins, but the end result may be as serious. The bite may or may not be excruciatingly painful. It is frequently not as severe as the agony usually suffered by the victim of a pit viper's bite. Swelling and edema are present, but they are slow in development and are never as severe as they are in a crotalid bite. Resultant symptomatology may develop within a half hour but usually takes several hours to reach a maximum, a fatal issue in mortal bites often not ensuing until 18-24 hours later. The patient rapidly becomes faint and gradually loess motor coordination. The sensorium is not greatly altered, although hyp- or hyperesthesia may occur in the region of the bite. The bulbar nuclei may become involved in a severe case, resulting in dysphagia. Due to motor paralysis, the patient may become flaccid with reflexes greatly impaired or absent. Gradual involvement of the respiratory center may eventually lead to a complete respiratory paralysis and death. Porges[4] points out that elapid venoms are extremely high in cholinesterase activity, whereas venoms of those vipers and pit vipers which are low in neurotoxic activity have almost no cholinesterase. Porges suggests that muscular paralysis of elapid bites may be due to the destruction of acetylcholine.

It should be emphasized that snake-bite morbidity is usually not as grave as that described above. Less than 10 per cent of treated crotalid bites are fatal. On the other hand, gravity of crotalid bites should not be minimized. One reason for the low mortality is the usual average size of the crotalid aggressor. Another is that more bites are recorded for the copperhead than for any other pit viper, and copperhead bites are only one per cent fatal. Untreated fatalities from large diamond-backs (5½-6 feet) are probably near 75 per cent and would be near 100 per cent were it not for the fact that a bite from a large animal does not necessitate a large expenditure of venom. It is difficult to estimate the death rate from coral snake bites as so few persons are bitten, but it is probably close to 25 per cent.

A few words should perhaps be said regarding bites of the Arizona Gila monster, the only poisonous lizard in the United States. The fatality rate, undoubtedly quite low, cannot be accurately estimated due to the poor records available. The venom is largely neurotoxic in action, and judging from the gravity of results seen in bitten laboratory animals it would be well to treat a Gila monster-bite with the same thoroughness as a snake-bite. The author was bitten through a sack in the fall of 1952. The wound was a quick slash through the pad of a finger, and the animal did not get a chance to hold on and chew. For a half hour there was local pain which was not excruciating. Little swelling resulted. Systemic effects were slight and consisted of nausea which was probably due to pain, tachycardia (not entirely due to pain, as it persisted at a rate of 120 after two hours), and a slight bilateral tinnitus occurring after an hour and lasting most of the day. The wound bled freely and still bled upon exertion of the finger three days later. Clotting time of the escaped blood was not prolonged.

ANATOMICAL CONSIDERATIONS OF VENOM SPREAD

Certain anatomical considerations of venom spread should be elucidated before treatment is discussed. Fillmore[6] points out that although the clinical behavior of the accompanying characteristic edema of crotalid bites makes it virtually certain that the venom is absorbed by way of the lymphatics, its circulation or movement through the tissues occurs primarily by way of the tissue spaces. He notes that this supposition is apparently justified by the observation that the edema readily ascends above known levels of communicating lymphatics. That is, in a bite situated on the fingers or hand, the edema, apparently limited to the skin, gradually ascends not only into the forearm, arm, and shoulder girdle, but to the chest wall as well. Fillmore points out that a review of topographical anatomy reveals that although the majority of lymphatics from the hand run to the axilla before perforating the deep fascia, these vessels do converge into the deep lymphatics at this level. An analogous situation is present in the lower limbs, except that communicating lymphatics have been demonstrated at the levels of the knee and thigh as well as in the inguinal region. The superficial lymphatics lie just beneath the skin in the superficial fascia, and continuity of tissue spaces in the skin is recognized.

TREATMENT

Pope's new concept: Clifford Pope, in 1950,[2] while stressing the necessity of removing the venom-laden lymph of crotalid snake bites through skin-deep incisions over the edematous tissue, cautions against incision over the fang marks, *i.e.*, at the site of the bite. Injection of colored venom beneath the skin of a dog demonstrated an instantaneous, rapid spread similar to that of ink when dropped on a blotter. In a finger or toe, the venom would immediately circle the digit and almost at once begin its journey proximally. Thus a cut into the site of the bite would not reach fictitious "pools" of venom but would largely be distal to the present location of the venom and in any case would reach only an insignificant quantity which could largely be removed later through a proximally placed incision. In a bite on the fleshy part of the arm or leg, it would not be as detrimental if the fang punctures were incised. Sometimes fang punctures cannot be differentiated from other tooth marks. However, on a hand or foot the necrotizing effects of the venom are marked, due to a paucity of tissue, and the additional trauma of a knife blade often aggravates or is responsible for a gangrenous issue. Any physician well acquainted with the sequelae of snake-bite can recall numerous cases of fingers maimed through necrosis of tendons, of fingers entirely lacking in subcutaneous tissue, or of digits which had to be amputated due to gangrene. The author was bittten over the proximal phalanx of the right thumb by a four-foot Western diamond-back soon after reading Pope's article. Somewhat flustered at the time and entirely forgetting Pope, he unfortunately instructed the physician who was with him at the time of the bite to incise over the fang marks. During the three days subsequently spent in the hospital, every refinement of treatment was executed. Despite this, the author was forced to suspend

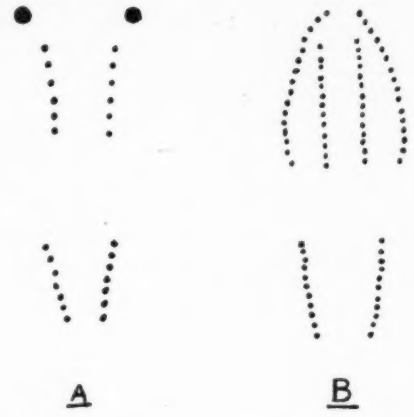
his surgical pursuits for nearly three months while waiting for a localized gangrenous ulcer to heal. Hypesthesia and considerable subcutaneous fibrosis remain to date, nearly three years later. The author definitely feels that the wound would have healed within two weeks if it had not been incised.

A. The tourniquet: Two functions are usually served by the tourniquet. One is to slow absorption of the venom and to allow its detoxification by the liver. The other function is to retain the venom in a portion of the body where it may be surgically removed from the subcutaneous tissue spaces. It should be remembered that localizing the venom in this manner is not an unmitigated blessing. Tissue necrosis is more severe locally as a result of sparing the body generally.

It is seldom that a patient is seen by a physician within a few minutes of being bitten by a snake. Fortunately the public has fairly well learned the importance of applying a tourniquet, so the first duty of the doctor usually consists of loosening the ligature — almost always applied too tightly for too long. Usual precautions should be taken with the ligature during subsequent treatment. If the physician recalls that only enough pressure need be applied to stop movement of the venom in the subcutaneous tissues, he may be justly inclined to leave it in place for considerably longer periods than he would for more customary tourniquet use. (The attendant applying the tourniquet may ignore the possibility of the venom having been deposited intravenously. It is rare, but if it does occur, the skills of an undertaker rather than a physician will probably be necessary.)

The tourniquet should be moved proximally ahead of the swelling. If the entire limb is swollen proximal to the tourniquet when the patient arrives, it is probably better to maintain it until program of incision has been carried out. This must be left to the judgment of the physician. If the snake is small and the patient husky, the doctor may well decide to dispense with surgery. If the patient arrives with no tourniquet soon after the bite, one should be applied immediately. If considerable time has elapsed with no tourniquet applied, and if the extremity is entirely swollen and systemic reaction is considerable, dispense with both the tourniquet and the scalpel.

B. Examination of pattern of snake-bite: After adjusting or applying the tourniquet, a brief history should be taken. The snake should be seen, if by some happy chance it was brought in with the patient, and the bite examined. Coral snake-bites cannot be easily analyzed, as these snakes chew the tissue, which action results in a haphazard series of lacerations. Pit vipers open their jaws widely but they do bite when they strike. The tooth marks of the lower jaw may be a considerable distance from those of the upper or, as in the case of a hand, on the opposite surface, giving the impression that two bites are present. Bite analysis may present considerable difficulty due to imperfect patterns. The following diagram, adapted from Pope and Perkins's Fig. 2,[7] represents tooth mark patterns as they would be if idealized:



A, western diamond-back rattlesnake
B, bull snake

C. Diagnostic value of pain: Considerable pain usually accompanies the bite of a poisonous snake, but occasionally it appears to be absent. Such absence may be attributed to a harmless snake-bite, to the relatively innocuous venom of a rearfanged snake, or to a tight tourniquet. Emotional factors must, of course, be evaluated.

D. Incision and suction: Extent of the physician's incision over the area of swelling must be tempered by his clinical judgment. If the snake was small and the damage probably slight, a few incisions forming a semicircle around the limb and over or anterior to the swelling may be sufficient. The individual incisions, placed one-half to one inch apart, would probably drain more efficiently if cruciate, but would be less mutilating if linear in either antero-posterior or sagittal directions. They need not be much more than one-eighth inch deep to reach the subcutaneous spaces. A massive injection by a large snake may necessitate several rings of incisions completely encircling the limb. New rings, if needed, should be placed about four to six inches proximal to the preceding ring and over the advancing edema.

Mild suction should be applied to the incisions by suction bulbs, breast pumps, or suction machines attached to such equipment as an ear speculum. Pope and Peterson[8] have experimental evidence involving dogs that alternating positive and negative pressure, such as might be obtained by use of a Pavex boot, is the most effective way to remove venom, although ordinary suction was efficacious. Combined tourniquet and suction may be usefully employed in severe cases for a dozen hours or more.

E. Supportive measures: Other than receiving tourniquet, incision, and suction treatment, the patient should be hospitalized for observation no matter how small the offending pit viper may have been. The patient should be blood typed before further hemolysis takes place. A urinalysis should be made and intravenous saline and blood administered if hemoglobinuria or albuminuria is present. Tetanus and gas gangrene antitoxins as well as antibiotics should be given. The Trendelenburg position should be employed if the patient is in shock, and utilization of the usual vasopressors may be necessary; use of a half-gram of $CaCl_2$ may lessen hemolysis. As much as 1000 cc. of citrated blood may be necessary every four to six hours. Barbituates and opiates may be used with caution. Minton[9] found that ACTH is of no benefit on experimental animals but feels that further observations would be desiarble. Draining wounds should be covered with moist, warm packs. Considerable caution should be exerted against use of excessive heat, as local tissues are anoxic and have no way to increase their blood supply if their metabolic demands are increased by the application of heat. The same deleterious effects will result from the use of cold, as will be shown in the next section. These statements cannot be overemphasized. Hot or cold applications that are well within toleration limits of normal tissue will result in extensive local blistering or more severe tissue necrosis if used on an extremity damaged by a pit viper-bite.

While most of these criteria may be modified for use in coral snake-bites, the emphasis should be placed on combatting CNS symptomatology. Use of such analeptics as metrazol and picrotoxin may be necessary. The supine position should be avoided as a paralysis of throat musculature may cause a fatal issue due to mucus obstruction. An airway may be employed in severe cases, and a drinker respirator or a closed pressure oxygen system may be instrumental in carrying the patient along until an otherwise lethal excess of venom is detoxified. It would be interesting to see how a patient would respond to an electrophrenic respirator. All these artificial systems should be more effective in treatment of coral snake neurotoxins than in treatment of bulbar poliomyelitis as there is apparently no permanent nerve injury from the neurotoxins.

In view of the fact that Porges (*cf.* page 6) has demonstrated a high cholinesterase activity in elapid venoms, it would be interesting to see what result could be obtained in the treatment of coral snake paralysis by the intramuscular injection of a test dose of 2 mgm. neostigmine methylsulfate and 0.6 mgm. of atropine sulfate. If any favorable response is shown, 2 mgm. more might be given in a half hour with a follow-up of 15 mgm. oral tablets as needed. It is certain that paralysis due to elapid bites resembles in pattern that due to a myasthenic crisis.

Emotional instability characterized by bursts of laughter or tears may persist for a year or longer, especially in the case of neurotoxic poisoning. Severe cramps in the affected extremity and, in case of an arm, in the involved pectoralis musculature may occur with decreasing frequency for two years following crotalid bites. Use of calcium gluconate may be helpful if these attacks become too frequent.

ANTIVENIN

Antivenin, as presently developed for pit viper-bites is largely if not entirely useless and may be highly dangerous. Proponents of the use of the substance recommend the injection of at least 75 cc. for severe bites. Seventy-five cc. of horse serum is a grim thing to contemplate, even if the patient is not sensitive. The author knows of several cases of bites by harmless snakes being converted into fatalities or near-fatalities by the use of antivenin. Ironically enough, the anaphalaxis is frequently attributed to the snake bite, with the feeling that the patient would have died had no antivenin been used.

Minton[9] feels that antivenin is of some use, although he does not give it a hardy endorsement. He found that white mice injected subcutaneously with pit viper venom and treated 15 to 20 minutes later with antivenin in doses of 2.5 to 5 ml./ kilo, were afforded some protection if they had received 1-2 LD/50 of venom. Those receiving 2.5-4 LD/50 of venom were not benefited. On the other hand, Peterson and Koivastik[10] found that the toxins of fresh European viper serum (*Vipera berus berus*) could not be neutralized by the serum of horses immunized against these toxins. The program was unfortunately not repeated with venom. The data are nevertheless significant, since European viper serum contains an antitoxin which is specific only for homologous venom.

This suggests that the toxin of viper serum is the same as that of viper venom.

Stickel[2] feels that pit viper antivenin will help neutralize the neurotoxins in pit viper venom but that the hemotoxins are not affected. Boquet,[11] on the other hand, in a discussion of parasympathetic activities of antivenins (p. 121), states that the hemorrhagins of various vipers and pit vipers were well neutralized by *Daboia* (viper) antivenin, whereas the neurotoxins were not at all neutralized. Boquet's whole work is a scholarly defense of antivenin. He attempts to define and correlate the conflicting evidence of other workers as well as present his own experimental data conducted *in vivo* as well as *vitro*. Unfortunately, he has no statistical analysis of actual cases of snakebite poisoning. In the whole literature devoted to the subject, there is apparently no statistical presentation of the value of antivenin for the treament of suake bite. There are a number of glowing testimonials, but they are not handled in a way that would satisfy even the most basic concepts of statistical analysis. At the present time, no physician should feel morally or legally obligated to use crotalid antivenin until a product has been developed which can guarantee results so improved that they offset the potential dangers of large amounts of injected horse serum.

There is somewhat better evidence that antivenins for neurotoxins are more effective. Due to the rarity of coral snakebites, it is unlikely that such serum would be available when needed. The serum is made by the Instituto Butantan, Sao Paulo, Brazil, and is often stocked by the larger zoos in this country. If available, it should be tried, although most of the specific effect will probably be lost, due to the fact that the antivenin is developed from coral snakes other than those occurring in this country.

The Poisonous Animals Research Laboratory at Tempe, Arizona, headed by H. L. Stahnke, has lately been publicizing the use of ice water as treatment for snake-bite as well as for the stings of scorpions. The "L-C Treatment," as described by Stahnke,[12] consists of "wrapping [a] string three or four times around the finger and then twisting it tightly, to the point of pain," placing a piece of ice on the site of the bite while "preparing a suitable vessel of crushed ice and water," and then immersing the bitten extremity into the iced water well beyond the location of the ligature. It is then suggested that the hand be kept in this mixture for "at least six hours . . . in order to prevent serious destruction of tissue. For a large rattlesnake, the patient may have to be treated for 14 to 20 hours." Stahnke wisely advises removal of his tight tourniquet after the hand, or other member, has been in the iced water for five minutes." Stahnke points out that the chemical action of the venom is halved for each 10° C. drop in temperature. He mentions that a "hand kept in iced-water will have its temperature dropped from the normal body temperature of 37° C. to about 4 — 7° C." Thus "the chemical activity at the site of the injection is practically nil." Stahnke substitutes iced-water for incision-and-suction in the belief that the latter is "not very effective . . . unnecessary and is only inviting more difficulties."

Stahnke also suggests the use of ethyl chloride in the field where ice is unavailable, recommending that the area of the bite or sting be sprayed to the point of frost formation and resprayed repeatedly as soon as the frost melts. In this manner "one tube can be made to last about thirty minutes." The only possible way for ethyl chloride to be effective at a depth of ½ inch (a depth of penetration easily achieved by the fang of a large snake) would be to freeze the surface layers solid. As mentioned earlier, there is enough anoxia and histolysis in the region of a bite without aggravating it by excessive cold, heat, or surgery. The tourniquet will achieve the same effect more safely and easily.

It is hardly necessary to remind a physician what happens when body tissue is lowered. to 4 — 7° C. for 14 to 20 hours Physicians who have served in Europe or in Korea are acquainted with immersion foot, resulting from prolonged expcsure in water as warm as 60° F. When the temperature of the skin falls to 25° C. (77° F.), tissue metabolism is slowed, and as the demand for oxygen remains greater than the slowed circulation can possibly supply, the area consequently becomes cyanotic.[13] At 15° C. (59° F.), tissue metabolism is markedly decreased, with consequent reduction of dissociation of oxyhemaglobin, resulting in a pink, well-oxygenated appearance to the skin. Available evidence indicates that tissue survival at this temperature is slight. Immersion extremity is characterized by a cold, anaesthetic member which changes abruptly in the hyperemic period to a hot extremity with intense burning and shooting pains. The affected extremity may be pale or cyanotic with diminished pulsations during vasospastic periods followed by swelling, blistering, redness, heat, echymosis, possible hemorrhage or gangrene, and possible secondary complications such as lymphangitis, cellulitis, or thrombophlebitis. This condition may be permanent or semi-permanent. If a patient suffering with Buerger's disease or Raynaud's syndrome should be

subjected to the Stahnke treatment, amputation would undoubtedly be necessary. It should be concluded that the "L-C treatment" of snake-bite should be rejected *in toto*. The use of tight ligatures is not only dangerous but is productive of much more pain than the incisions that Stahnke deplores. Iced water is not only immediately painful but leaves sequelae which may result in amputation or a permanently physiologically deranged extremity.

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REFERENCES

(1) H. K. Gloyd, Snake poisoning in the United States: A review of present knowledge; Chicago Med. Soc., Bull., Jackson Park Branch, 15 (7): 3, 5, 7, 9, 11; 15 (8): 13, 15, 17, 1938.
(2) William H. Stickel, Venomous snakes of the United States and treatment of their bites; Wildlife Leaflet of the U. S. Fish and Wildlife Service, 339: 1-29, June 1952.
(3) Clifford H. Pope, The snake-bite problem; Chicago Natural History Bulletin, 21 (6): 6-7, June 1950.
(4) Nandor Porges, Snake venoms, their biochemistry and mode of action; Science, 117: 3029, January 16, 1953.
(5) Forest H. Stanley, The present rationale in the treatment of poisonous snake bites; Weekly Bulletin of the St. Louis Medical Society, 44: 548, June 23, 1939.
(6) R. S. Filmore, The early local treatment of snake bite; Texas State Journal of Medicine, 37: 311, August 1941.
(7) Clifford H. Pope and R. Marlin Perkins, Differences in the patterns of bites of venomous and of harmless snakes; Archives of Surgery, 49: 331-336, November 1944.
(8) Clifford H. Pope and L. W. Peterson, Treatment of poisoning with rattlesnake venom; *Ibid.*, 53: 564-569, November 1946.
(9) Sherman A. Minton, Snake-bite in the Midwestern region; Quarterly Bulletin, Indiana University Medical Center, 14 (2): 1-4, 1952.
(10) H. Peterson and T. Koivastik, The toxicity of serum of the European viper (*Vipera berus berus*) and its antitoxic properties relative to homologous and heterologous snake poisons; Zeitschrift fur Immunitatsforschung und experimentalle Therapie, 102: 324, December 10 ,1942.
(11) P. Bouquet, Venins de serpents et antivenins; Collection de l'institut Pasteur, Editions Medicales Flammarion: 1 - 157, 1948.
(12) Herbert L. Stahnke, The L-C treatment of venomous bites or stings; Arizona Highways, 29 (2): 35, February 1953. Reprinted from American Journal of Tropical Medicine and Hygiene.
(13) Many authors, Handbook of Medical Management: University Medical Publishers, Palo Alto, California, 1st. ed.: 418-420, 1949.

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RECURRENT IMPLANTATION CYSTS OF THE OVARY

By A. H. Follingstad, M. D., Albuquerque, N. M.

The two cases reported herein are of interest both from a diagnostic and a therapeutic point of view. A rather thorough search of American medical literature, including standard gynecological texts, and published articles, failed to reveal any mention of recurrent aberrant or implantation cysts of the ovary. A review of foreign medical literature was rewarded by the finding of one article describing a case similar to the two reported here. C. Roman[1] reporting in a French journal, described a case of cystic degeneration of "ovarian debris" following an incomplete oophorectomy which was treated successfully by irradiation.

These cysts are man-made and follow incomplete or careless surgery on the uterine adnexae. Despite the paucity of information on this pathological entity, there must be many similar cases seen yearly which present a therapeutic problem to the practitioner. It is hoped this article will be useful in creating an awareness of this type of cystic disease of the female pelvis and in pointing out a simple treatment for the condition.

CASE ONE

Mrs. L. J. — age 37 was first seen in Sept. 1948 with the complaint of recurrent pain in the lower abdomen and metrorraghia. Her past history disclosed four abdominal operations, two of which were gynecological in character. One tube and ovary were presumably removed during one operation, and subsequently the remaining tube and ovary with a uterine fibroid were supposed to have been removed. Pelvic examination revealed an orange sized cystic mass occupying the left adnexal region and cul de sac, a normal sized uterus and a small mass on the right thought to be an ovary.

The patient was operated upon in Oct. 1948. Multiple and dense generalized adhesions were found and separated. The uterus was the site of multiple fibromata with a right hydro-salpinx and cystic degeneration of the right ovary. No tube or ovary were found on the left, but an orange sized serous cyst was shelled out in toto from the left broad ligament. A subtotal hysterectomy and right salpingoophorectomy were performed.

The pathological report was as follows:

"The gross specimen consists of the uterine fundus 5 cms. in diameter and 4 cms. in length. This shows one subserous fibroid 8 mm. in diameter. Gross section shows an intramural fibroid 1 cm. in diameter. The tube on one side is occluded, and shows a hydro-salpinx through the occluded fimbria. The ovary on the opposite side measures 3 cms. in diameter and shows three follicular cysts 5 to 8 mm. in diameter and a hemorrhagic lutein cyst 12 mm. in diameter. There is a portion of the tube attached to this area."

"There is an isolated cyst 5 cms. in diameter which contains a sero-sanguinous fluid and a partially organized blood clot."

"Microscopic section made through one tube shows a rather rough granular surface."

Diagnosis: "Multiple fibroids of uterus. Cystic degeneration of ovary. Hydro-salpinx."

In March 1949, five months following her operation, the patient had spotting for one day. Examination disclosed no evidence of the usual atrophy of menopause, although no ovarian tissue should have been present to produce an estrogenic stimulation. A 5 cm. cystic mass was palpated in the L. adnexal area, and the spotting was presumed to be coming from a small area of supra-cervical endometrial tissue inadvertently left during the subtotal hysterectomy.

In October 1950, one year later, the cystic mass had grown to the size of a baseball, and continued estrogenic activity from some source was evident. The patient was again subjected to surgery, and pelvic exploration revealed a large cyst occupying the left lateral pelvis. This again was shelled out in toto by blunt and sharp dissection and inspection revealed the cavity to be formed by the lateral pelvic wall and the left side of the rectum and upper vagina. Any attempt to extirpate the lining of this cavity by surgical excision would, obviously, seriously jeopardize vital pelvic structures such as ureter and rectum, and the abdomen was closed without further surgery.

The pathological report on this cyst was:

"The gross specimen consists of a 2.5 cm. thin walled cyst which appears collapsed. Representative section of the wall is taken for microscopic study."

"Microscopic shows the tissue to consist of a group of cysts one of which is lined by an undulating luteinized wall. A smaller cyst shows a marginal lining consisting of an atrophic layer of granulosal cells. In addition there is diffuse infiltration of part of the tissue by large numbers of lymphocytes and large mononuclear phagocytes, many of which contain hemosidern and are concentrated about the wall of a third cyst. The

two larger cysts appear to be lined by a layer of granulation tissue, and no epithelial or endometrial elements are demonstrable."

Diagnosis: "Follicular, lutein and probably endometrial cysts of left ovary."

The true nature of this recurrent cyst was finally evident. A bit of ovarian tissue had been buried in the left broad ligament during one of the previous gynecological procedures. This had formed a true follicular or thecal cyst, consisting of a cavity bounded by the lateral pelvic wall, rectum and vagina and lined by estrogen secreting granulosa cells. Removal of the one cyst would inevitably be followed by the formation of another.

Recurrence of a cystic mass on the left side was predicted and was palpably evident 6 months later. With final knowledge of the exact nature of this recurrent lesion, deep X-ray therapy was given to the left side of the pelvis. Regression of the cystic mass occurred and with this, menopausal symptoms ensued. Up to date, there have been no findings of recurrence. The irradiation apparently completely destroyed the aberrant ovarian tissue.

CASE TWO

Mrs. H. G., age 24, was first seen in August 1950, with the complaint of frequent menstral periods (every 21-23 days) associated with severe premenstral tension and marked dysmenorrhea. In 1945 a laparatomy had been performed at an Osteopathic Hospital, at which time both tubes and ovaries were supposed to have been removed. Pelvic examination revealed a rather small uterus with an ill defined fullness and tenderness in the left adnexal region. The patient was followed for one year under various types of conservative management. During this time menometrorrhagia developed, dysmenorrhea increased in severity with pain radiating to the rectum and the mass in the left adnexal area increased to the size of an orange.

An exploratory laparotomy performed in Oct. 1951, revealed the following findings: The right tube and ovary were absent. A small portion of the proximal end of the left tube was present, but no ovary was found. A large cystic mass occupied the left broad ligament. The broad ligament was opened, the cyst dissected out in toto and the defect in the broad ligament was closed.

The pathological report was as follows:

"Gross specimen consists of a hemorrhagic ovarian cyst which measures approximately 8 cms. in greatest diameter and presents an irregular hemorrhagic internal surface. There are no papillar excrescences on the external surface and except for areas of hemorrhage, the internal surface appears smooth. Multiple sections are taken from the wall for microscopic study."

"Microscopic shows the cyst wall to be formed by atrophic ovarian stroma containing several corpora albicantia and one medium sized follicular cyst. There is focal interstitial hemorrhage within the cyst wall. The internal lining of the cyst is formed by an atrophic zone of condensed ovarian stroma with no demonstrable epithelial elements. There is no evidence of endometrosis or malignancy."

Diagnosis: "Follicular cyst, ovary large."

One month following her operation the patient had a 3 day menstral flow despite the apparent total absence of ovarian tissue. Two months later another menstral flow occured and a cystic mass again could be palpated in the left side of the pelvis. The lesson learned from the previous case made the diagnosis easy this time and precluded any further pelvic explorations. The early unwarranted and unskillful gynecological procedure had buried a remnant of ovarian tissue in the broad ligament, and this bit of ovary, stimulated by the removal of the opposite ovary formed a thecal lined cyst similar to that of the previously described case. The patient was given deep X-ray therapy to the left pelvis and the expected result followed. Permanent regression of the cystic mass was obtained and menopausal symptoms requiring estrogen therapy ensued. To this date, no evidence of recurrence of the cystic mass has been noted.

SUMMARY AND CONCLUSIONS

Two case reports are given describing an unusual type of recurrent cystic disease of the female pelvis caused by unskillful or incomplete surgery on the uterine adnexae. A review of the literature indicates that this condition is not well known and little understood. In most instances, complete surgical exturpation of the lesion would be impossible or hazardous due to the close proximity of vital pelvic structures, but if the condition is recognized, a simple and safe method of treatment is available, namely, irradiation.

1. C. Roman, Lyon Chirugical, Vol. 41, No. 6 — Nov.-Dec. 1946.

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